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THE INFLUENCE OF THE VENÆ COMITES ON THE PULSE TRACING, WITH SPECIAL REFERENCE TO VALSALVA'S EXPERIMENT, AND DICROTISM; A NOTE ON ANACROTISM.

By THOMAS LEWIS, D.Sc., M.B., B.S. (Twenty-eight Figures in the Text.)

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THE INFLUENCE OF THE VENÆ COMITES ON THE PULSE TRACING, WITH SPECIAL REFERENCE TO VALSALVA'S EXPERIMENT, AND DICROTISM; A NOTE ON ANACROTISM.

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THE literature dealing with the effects of respiratory movements on the circulation is very extensive. As an outcome of the researches of Einbrodt[5], Funke and Latschenberger[7], Kowalewsky[11] and numerous other observers on animals, and the experiments of Marey[16], Riegel[19], v. Basch[1], Wertheimer and Meyer[26] and others on man, certain conclusions have been reached. The work was summed up by Tigerstedt[25] in his text book of 1893. The prevailing opinion at the present time is that inspiration causes, after a preliminary fall, a rise of blood-pressure; and that conversely with expiration there is first a slight rise of pressure, but that the main effect is a fall. There have been difficulties in harmonising some of the observations obtained from man and animals, and this has been particularly the case in the study of the effects of increased and decreased intrathoracic pressure; thus while increased intrathoracic pressure is generally recognised as giving a fall of blood-pressure in animals [7], [11], [18], [27], yet it is a well known phenomenon that there is a rise in the curve of the sphygmograph tracing, taken during Valsalva's experiment[16]. Moreover it is stated by many writers that this rise is accompanied by signs of diminished arterial tension[10], for it is accompanied by diminished excursion of the pulse beat, and increased dicrotism. In the tracings given by some authors[16], there is also an increased frequency of heart beat. In 1880 the position was reviewed by Rollett[20] and its difficulties were shown. In 1890 Roy and Adami[21] discussed the causation of the rise and attributed it to rise of arterial pressure. They came to the conclusion (cp. [13] also), that "the depth of the dicrotic notch is increased by any cause which diminishes the volume of blood thrown out by the ventricle at each contraction; and also by any cause which, *cæteris paribus*,

raises the pressure within the systemic arteries." Since this publication, Hill, Barnard and Sequeira[10] have reinvestigated the question and conclude that "a rise of intrathoracic pressure raises venous pressure and lowers arterial tension. The combined effect of these variations leads to the elevation of the line of the sphygmograph curve." Briefly, they attribute the rise to a swelling of the venæ comites accompanying the radial artery; their experiments were conducted on man and animals with the same instrument, whereby one group of fallacies was eliminated, and the evidence which they adduce appears at first sight conclusive. The experiments upon which this communication is based have been carried out entirely on man; the instrument used for recording the pulsations of the radial artery has been that used by the last-mentioned observers, namely Dudgeon's Sphygmograph. For the most part the experiments have been conducted on four individuals, in age from 19 to 28 years. In none of these four subjects was there reason to suspect any abnormality of the cardio-vascular system; the systolic blood-pressure ranged on the average from 120 to 135 mm. Hg. The pulse tracings which they gave are typical of normal curves. The other instruments which have been employed are Mackenzie's polygraph[14], Ludwig's kymograph[1], and Dr C. J. Martin's modification of the Riva-Rocci sphygmomanometer[2]. In taking plethysmographic records it has been found convenient to use the armlet of the Riva-Rocci filled with water and connected by a water system in rigid tubing, to a Schäfers piston recorder.

Early in the course of the investigation it became apparent that the method of fixing the Dudgeon's Sphygmograph is open to a serious objection. The instrument requires no detailed description here, but it is necessary to state that it is fixed to the limb under observation by a band encircling that member, and that the record is taken from a small steel pad, riding between two lateral standards. For experiments on venæ comites this arrangement is an impossible one, for the fixing band superimposes on the pulse curve, which in the main is an index of pressures, a plethysmographic effect. A rise in the curve accompanies the slightest added tension to the band; a rise consequently results from any increased volume of the limb, whether it has its origin

[1] The experiments necessitating the employment of this instrument were performed at University College, Cardiff, and my best thanks are due to Prof. Haycraft for the use he has given me of his apparatus. The tracings shown in this paper are all considerably reduced in size.

[2] As supplied by Hawksley, London.

in venous or arterial plethora. In the following experiments the band fixation has been replaced by a rigid extension, fixed above to the upright rod carrying the lever mechanism, and falling below on either side of the wrist to a small hook, to which any desired weight may be attached. A small bucket containing shot has been found convenient. The apparatus as first used is shown in Fig. 1, but latterly it has been found more convenient to suspend the weight from a single curved bar counterweighted.

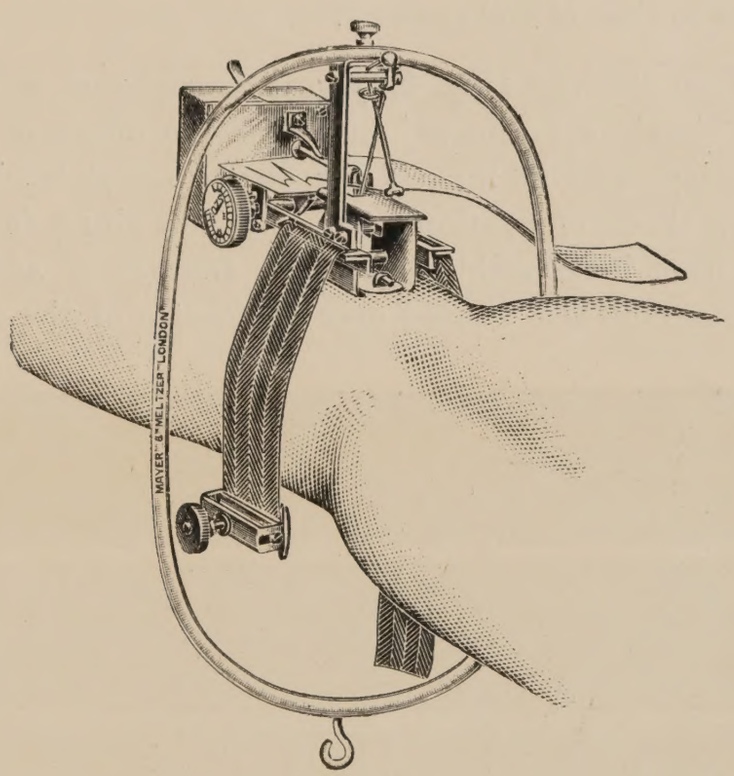


Fig. 1.

In the first instance a comparison was instituted between the Dudgeon's Sphygmograph, with the old and new method of fixation. Adjustment is as simple in the one case as in the other, but there is greater precision with the new method. Briefly it may be stated, as a result of a considerable experience of both instruments, that the new method of fixation has great advantages, chief amongst which is the elimination of the effects of slight muscular movements. The general shape of a pulse beat curve is practically identical in most cases, when obtained by the two methods, but with the band fixation the amplitude is frequently greater. This difference in excursion is attributed to the added plethysmographic effect and in some pulses is marked. The old method of fixation frequently gives an indication of pulse beats when the pad is off the artery. In testing the effect of venous congestion on

the pulse a method is adopted which is believed to be a very exact one. The Riva-Rocci armlet is placed on the upper arm and the pressure raised to any desired height by pumping air into it. The pressure in the veins accumulates until it is of sufficient height to force the block, a pressure which is equal, with a small error, to the pressure in the armlet. In no case is the venous pressure allowed to rise to diastolic pressure; consequently the tracing is little affected by direct pressure on the brachial artery. The diastolic pressures are estimated by a method similar to that of Erlanger[6].

Exp. 1. Four tracings (Fig. 2, typical examples of 40 tracings) are given from a simple experiment, and show the effect of a release of venous congestion, from 60 mm. of Hg, on the radial tracing, with the old and new methods of fixation. The arm was slung (as in all these experiments) from inextensible tapes. In tracings A and B, the pad of the sphygmograph was resting over the centre of the radial artery,

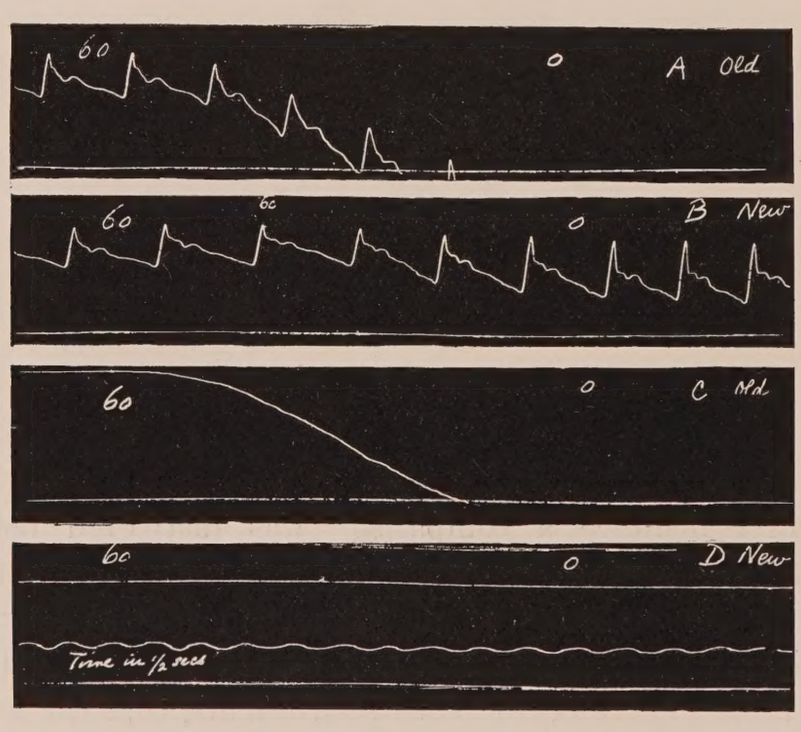


Fig. 2.

and the tracings were maximal. In tracings C and D control experiments were made from the front of the wrist[1]. While tracings A and B were taken, the extension was altered but the pad remained in place: so also in the case of tracings C and D. In each case the venous pressure was reduced by detaching the tube entering the armlet. On

[1] At a point where no veins are apparent.

comparing the tracings the effect of volume change in A and C is obvious. The effect of the congestion with the new fixation is seen to be only slight, the beats fall slightly, while the control, (D), is a straight line. The time tracing in D applies to the whole experiment. The result of this and similar experiments has justified the discarding of the band fixation and in the tracings which follow the weight extension has been used.

A priori, there are certain facts which weaken the evidence produced to support the theory of venous interference in Valsalva's experiment[1]. The capacity of the veins of the body is well recognised. Plethora can be induced to such an extent by infusing [24], as to suggest the impossibility of much rise in venous pressure in an experiment, necessarily so short as Valsalva's. The actual measurement of venous pressure is very difficult. The method adopted in Exp. 1 provides a means of measuring venous congestion in mm. Hg very approximately, (cp. Exp. 2), in any individual, but the method takes time, and it is impossible by its use to measure venous pressure at any given instant. In Valsalva's experiment a tendency to venous engorgement is sometimes visible, but it is the exception rather than the rule, and is never great. A photograph, Fig. 3 A, is given of an arm at the height of a Valsalva's experiment

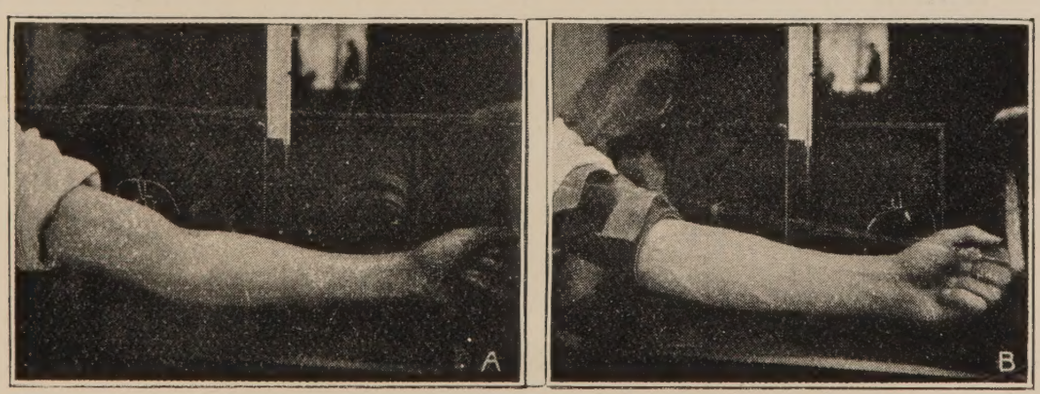


Fig. 3.

lasting 9 secs. An indication of the intrathoracic pressure was obtained by allowing the subject of experiment to blow into a manometer, and in this case was equivalent to 70 mm. of Hg[2]. No venous congestion is visible; yet the control photograph, Fig. 3 B, taken from the same arm with the venous pressure raised to 40 mm. Hg, shows marked distension of the veins. By employing different venous engorgements it can be shown

[1] Cp. François Frank, *Physiol. Expér. Marey.* 1877, t. II. p. 57.

[2] I am aware that the blowing pressure is not equivalent to intrathoracic pressure.

that when a deep inspiration is followed by forcible expiration with the glottis closed, the venous pressure accumulating in the arm is certainly far less than 20 mm. Hg if the arm is horizontal, even with very forcible and prolonged expiration. In those instances where investigators have occluded all the veins of the limb and the pressure rises to static arterial pressure, the comparison drawn between the effect of the venous pressure on the tracing in this and Valsalva's experiment is not a fair one. Another point of considerable importance, which has so far only been touched upon, is the rate of rise of venous pressure. Ten seconds is a fairly wide limit for the duration of a chest compression, yet in this short time interval sufficient blood has to enter the veins from the capillaries to engorge them in their whole length, to a pressure necessary to affect the tracing. The next experiments have been devised to give some idea of the time taken for venous engorgement to occur in the limit. It must be remembered that as in these experiments the block is situated at the lower part of the upper arm and not at the outlet of the veins in the chest, the accumulation of pressure in the venæ comites will be far greater in rate than in Valsalva's experiment. Also it must follow that whatever be the pressure of the block, venous engorgement will proceed at the same rate. Further it is of importance to note that in these experiments the whole venous circulation of the limb is obstructed at the same time and approximately to the same extent. In Hill and Barnard's experiments[10] all the veins of the limb were first occluded by ligature, leaving only the venæ comites. These were subsequently compressed. There is every reason to suppose a preliminary engorgement of the limb before the commencement of the experiment in this case.

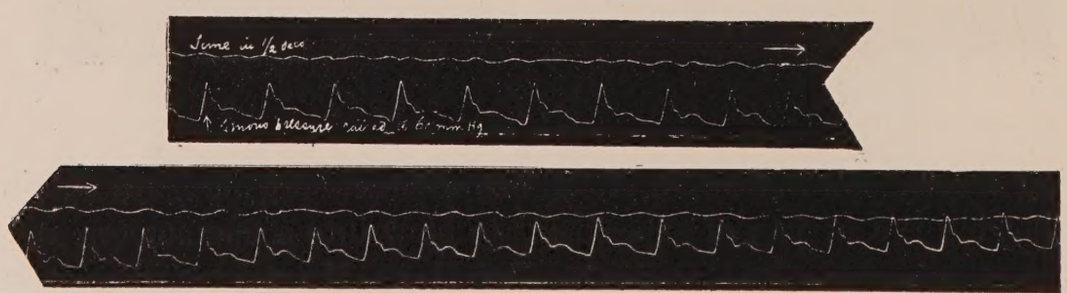


Fig. 4.

Exp. 2. (*a*) The essential part of a tracing is shown in Fig. 4, one of 50 similar tracings. The arrow shows the point at which a block of 60 mm. Hg was thrown upon the veins of the upper arm. During the first 12 seconds the tracing is unaffected; there is a gradual

and steady rise during the remaining 11 seconds shown (which was continued for another 20 seconds). The total duration of increasing congestion was consequently in this case 48 secs.

(*b*) The same effect is shown plethysmographically in Fig. 5, one of 10 experiments at different pressures. Venous congestion was produced to 40 mm. Hg by the same method as that used in 2 (*a*), and was recorded by the Riva-Rocci armlet used as a plethysmograph, a method which has been found to answer well in taking volume records from the human arm. The tracing shows a delay of $7\frac{1}{2}$ secs., and a rise occupying 21 seconds. The tracing is from another individual. In Exp. 2 (*a*) and 2 (*b*) the delay in the rise is attributed to the time interval taken for the venous pressure to rise in the veins, from the block down, to a height sufficient to overcome the original pressure exerted by the recording instrument, sphygmograph pad or plethysmograph, on the tissues underlying the skin. Considering the sphygmograph pad, the

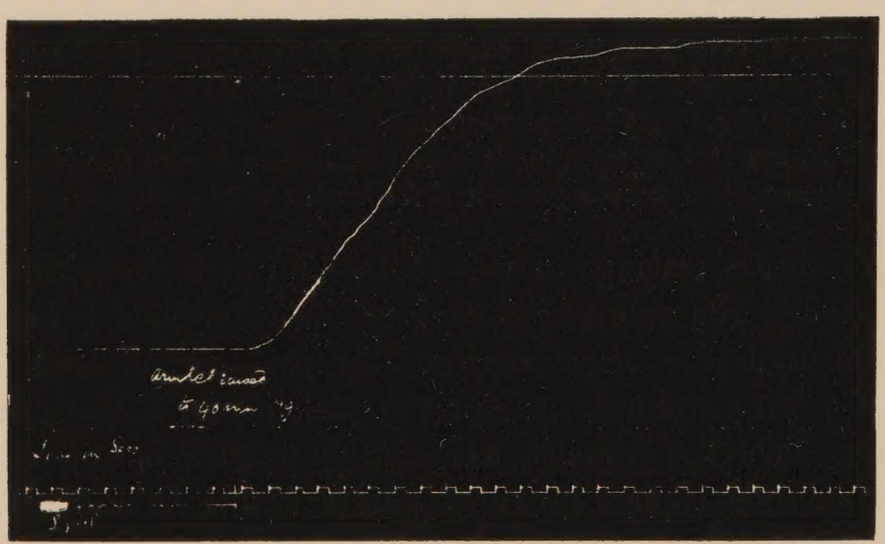


Fig. 5.

pressure which it exerts on the skin is counteracted by two pressures; the resistance of the tissues, in which the skin probably plays the chief part, and the pressure exerted by the blood in the artery upon the pad. The latter is equivalent to the pressure exerted by the pad on the artery, and it is not until the pressure in the veins has risen to this point, that the height of the tracing will be influenced. For up to this pressure any veins pressed upon by the pad will be collapsed. The pressures existing during the taking of a tracing are diagrammatically represented in Fig. 6. The experiment 2 (*a*) is of service as it enables the observer to estimate, within certain limits, the pressure exerted on the

radial artery by the sphygmograph pad in any given experiment. This pressure will vary constantly with every application of the instrument. If reference be made to Fig 2 B it will be apparent that the diastolic portion of the tracing is first affected, giving a greater amplitude of excursion. The application of this method will be discussed more fully in a future communication[1]; here it may simply be stated that it is

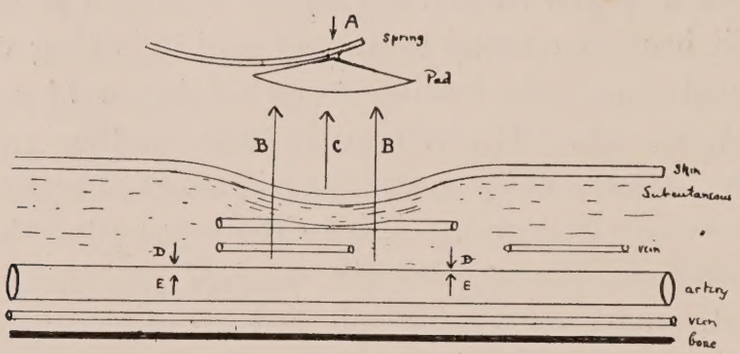


Fig. 6. *A* represents the pressure of the pad on the skin. *B*, *B* represent the resultants of two pressures, *D* and *E*, where *E* represents the blood-pressure in the artery, and *D* the pressure of arterial wall and surrounding tissues on the blood column. Under ordinary circumstances they are equal, with indentation *D* is decreased, and *B* increased. *C* represents the resultant pressure, due to tissue resistance, pressing on the pad. At any instant when the levers are stationary $A = B + C$. When the venous pressure increases beyond the pressure *B* the tracing will be affected.

possible to obtain tracings of this character in which diastole is alone affected, though the process is laborious. The method is based on the supposition that the space enclosed by artery, pad and standards (Fig. 7) is a fairly uniform one, for the pad is very little broader than the artery. That the pressure is absolutely uniform is improbable; and that the method should prove of clinical value is unlikely, but for careful experiment it should give far more accurate results than an estimation of pressures exerted by the pad on the skin[2]. The subject is mentioned here, for in comparative experiments on cats and dogs, where the vessels of a limb are laid bare, the artery comes into contact with a limited surface of the pad, the veins occupying the remaining space (Fig. 8). Moreover there are no overlying tissues to distribute the pressure. Consequently *any* rise of venous pressure will increase the average pressure on the pad and raise the lever on the smoked paper. It is for this reason that the present experiments have been conducted on man only, as comparative work on man and animals must be

[1] *Practitioner*, 1906.

[2] As roughly indicated by the Roman figures on the circular disc of the sphygmograph.

misleading. The experiments of Hill, Barnard and Sequeira are open to this objection; the rises in the tracings given show no delay.

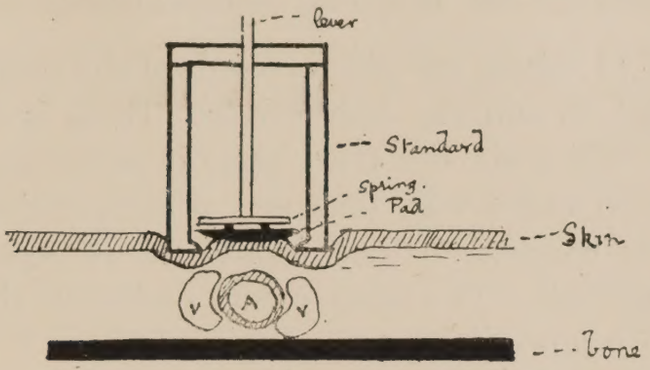


Fig. 7.

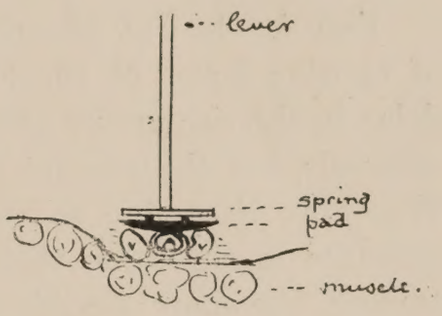


Fig 8.

Fig. 7. Diagram of sphygmograph applied to radial artery.

Fig. 8. Diagram of sphygmograph applied to vessels of dog.

Exp. 3. Fig. 9 (one of 86 other tracings) is a typical example of a pulse record during Valsalva's experiment. The blowing pressure in this case was 60 mm. Hg. The rise in the curve shows no delay and reaches a maximum in about $3\frac{1}{2}$ secs. Dicrotism is well marked at the 5th sec. A control tracing was then taken (Fig. 4), without moving the sphygmograph; venous congestion was produced to 60 mm. Hg. The delay in the venous rise was, as has been seen, 12 seconds. In this experiment it is certain that venous congestion had nothing to do with the initial rise, and moreover, what is of far more importance, it had no influence on those dicrotic beats which are raised. In this tracing there

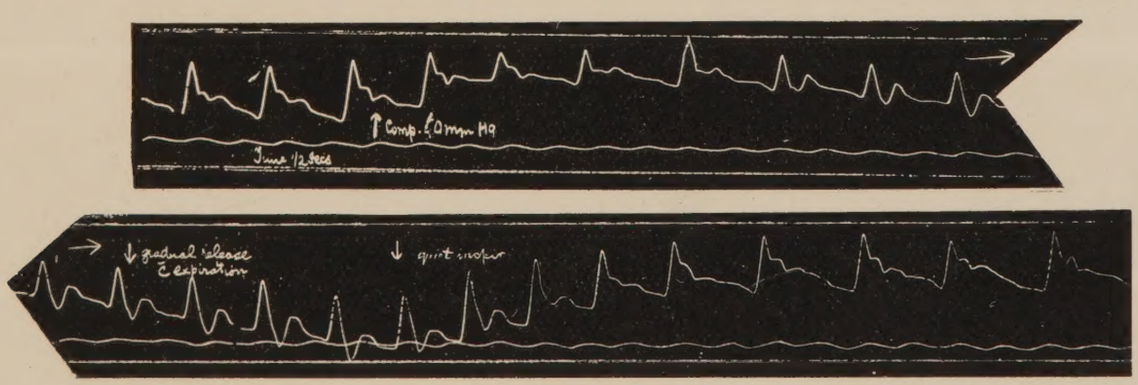


Fig. 9.

is a falling away of the curve before release of the chest pressure, and this is a fairly constant phenomenon; it is not so marked in tracings taken with the band fixation (cp. [16], [23]). It is notable that the beats are well marked throughout; the decrease in excursion is insufficient to indicate a decline in the force of the heart beat, and those dicrotic beats

which occur above a mean line are further apart than the normal beats; (this is opposed to the statements of Marey[16], though his tracings show an increased distance between the first few dicrotic beats).

Exp. 4. In Fig. 10, one of 11 others, are given records of the result of blowing forces of 40, 60 and 80 mm. Hg respectively. There is no delay in the rise in any case[1]. The main rise takes place in equal time intervals, yet the amount of rise varies with the force of compression. Were the rises due to venous congestion, they should be of equal amounts in the same time intervals. Dicrotism occurs earlier in the tracing, the more forcible the compression.

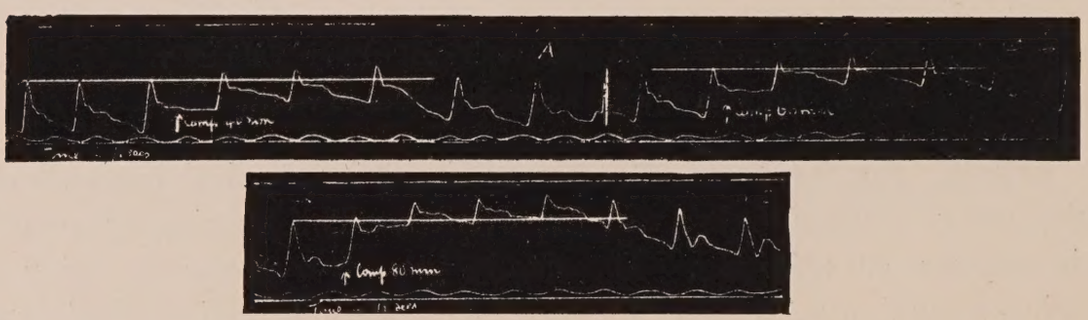
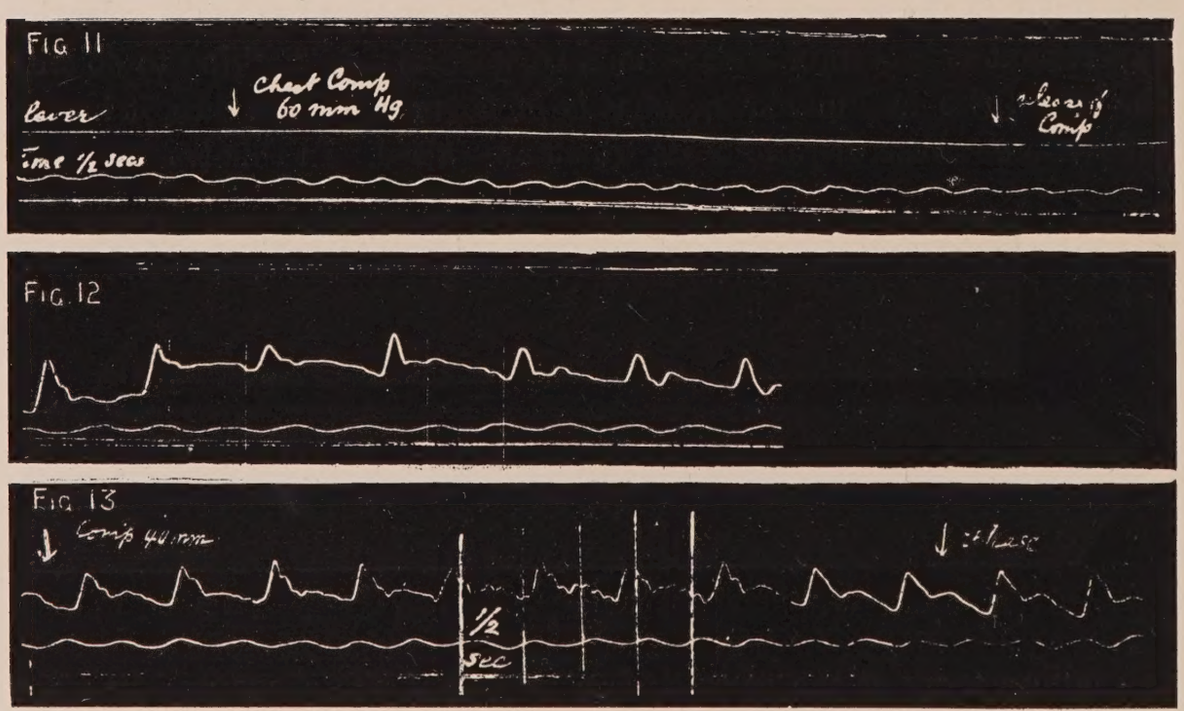


Fig. 10.



Figs. 11—13.

[1] In many tracings given, arrows have been filled in subsequent to the experiment. Their positions are based on experiments with a polygraph.

Exp 5. In this experiment (Fig. 11, one of 20), the pad was resting lightly on the superficial veins of the forearm. Compression of the chest as a rule gives no rise in the line of tracing under these conditions. A slight rise has occasionally been seen in very prolonged compressions, and with the pad resting as lightly as possible.

Exp. 6. In many of the curves, small oscillations at about 10 per sec. are superimposed on the pulse curves, as shown in Fig. 12. They occur when the muscular effort is great, and are probably not conducted through the tissues of the arm, for controls from indifferent spots do not show them to nearly the same extent. There is reason to believe that they are transmitted from the chest as pressure oscillations in the arterial blood. They could not be transmitted by the veins. Usually they are better marked in compressions following deep expiration, when the respiratory muscles are working at a disadvantage (Fig. 13).

Exp. 7. In Fig. 14, (A and B), two of 39 tracings, the first rise in Valsalva's experiment is interrupted by occlusion of the brachial artery, when dicrotism appears. The line of tracing (Fig. 14 A) falls promptly and rises promptly on release. The result indicates that the lever is supported by arterial rather than venous pressure. When the occlusion of the artery takes place towards the end of the curve, when it is falling, the drop is not so marked, (Fig. 14 B). Some evidence has already been given to show that the last few beats of a prolonged compression may be supported by venous congestion. This will be particularly the case when pad pressure is light. There is sometimes slight lowering of the line when compression of the chest is released before the release of the artery (Fig. 16). There is however always the possibility that the brachial occlusion is not complete and there is an indication of beats in the compression line in Fig. 16. A diminished fall during the chest compression is the rule in tracings from the dorsalis pedis, with the foot somewhat dependent, and there is also some

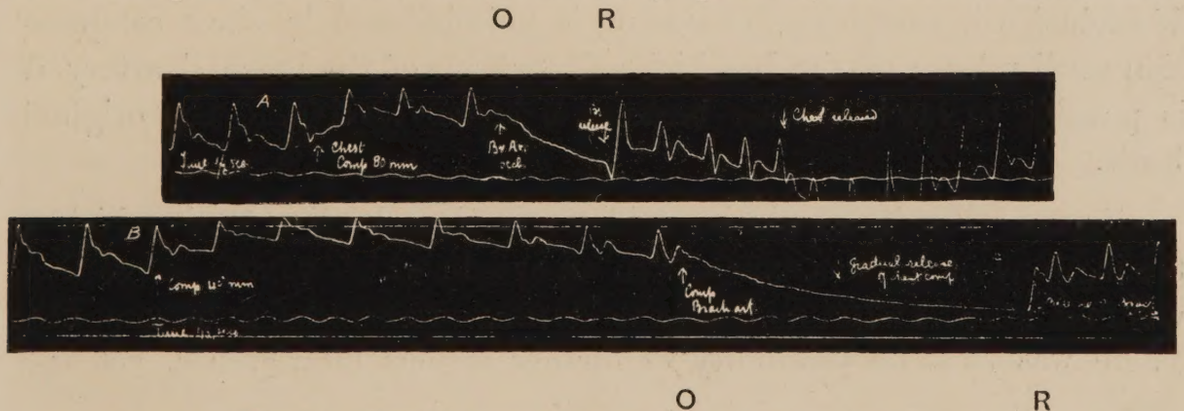
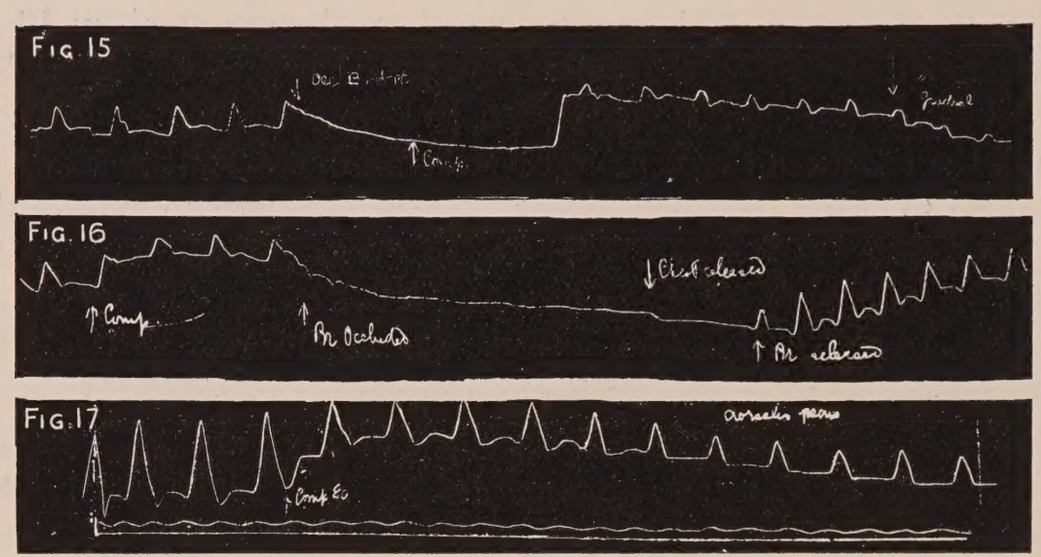


Fig. 14. Brachial artery occluded at *O*, released at *R*.

modification of the diastolic phases of the beats (Fig. 17)[1]. The disagreement of these tracings with that given by Hill, Barnard and Sequeira[10] is probably due to the fact that in these experiments weight extension has been used to fix the instrument.

O C



Figs. 15—17.

Exp. 8 is the converse of the last. The tracing, Fig. 15, one of 30, was interrupted by a brachial occlusion *O*, followed by chest compression *C*. If occlusion is complete, the tracing is uninfluenced by chest compression until release of the brachial. On release of the brachial artery it is possible to catch dicrotic beats at a higher level than the original beats.

Exp. 9. Finally conclusive evidence is given by Fig. 18, in which a tracing, one of 52, was taken from an arm in which venous congestion was first raised to 70 mm. Hg; the blowing pressure being 60 mm. Hg. There was thus no possibility of further venous interference, yet the

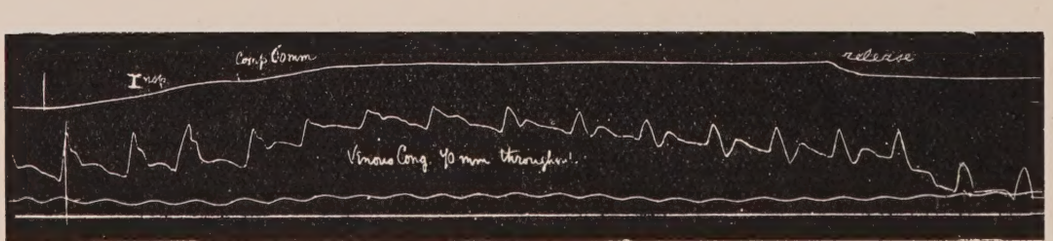


Fig. 18.

[1] The tracing has very much the same characters as that taken from the radial, showing that the pressure on the abdominal aorta has only slight effect on the limb circulation.

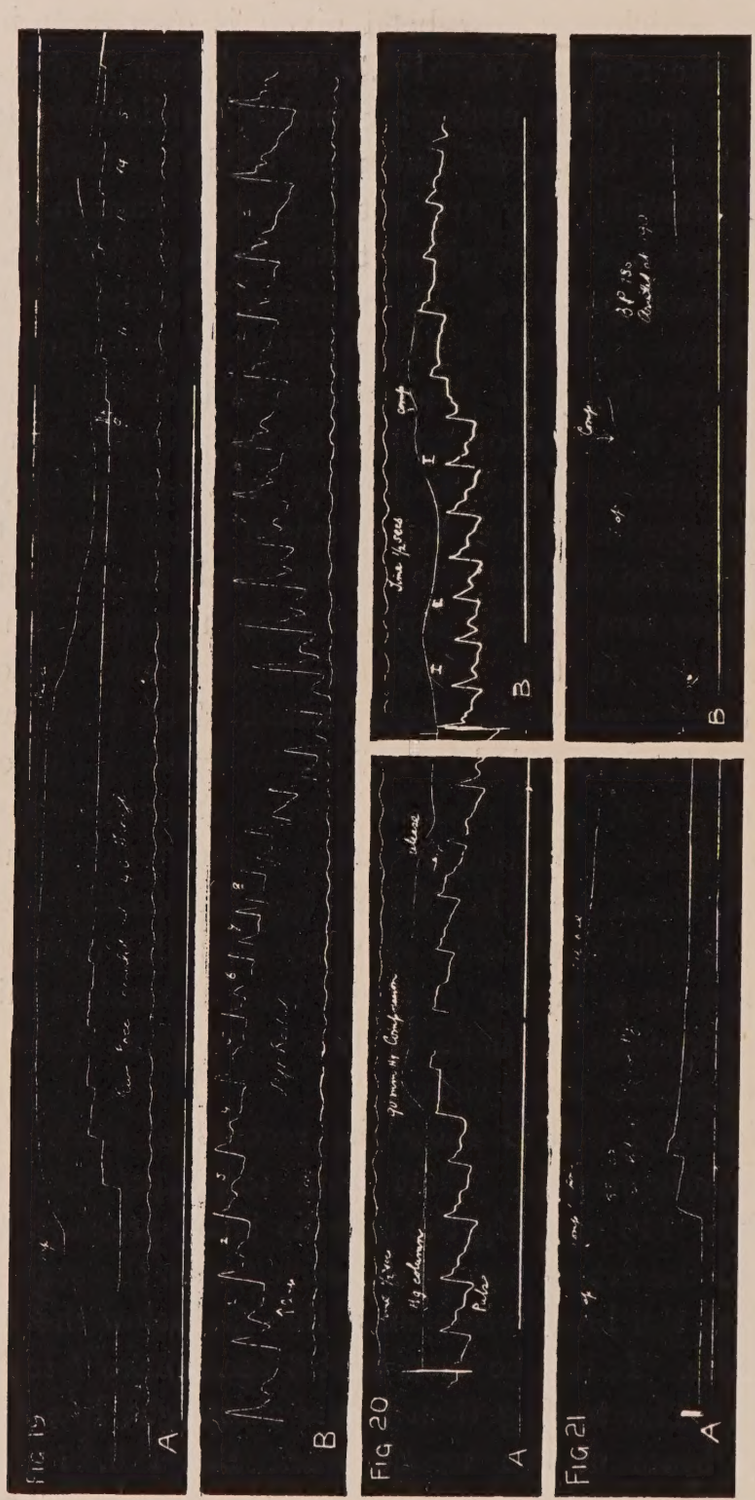
tracing is typical throughout. The tracing shows a respiratory line taken with the polygraph. Again, in experiments of this kind, if very light pad pressure is used, the fall is interfered with by the original venous congestion, and the diastolic phases of the beats may be obscured.

The evidence which has now been given is overwhelming against the theory that the rise in Valsalva's experiment is due to venous congestion. It is true that under favourable conditions the last few dicrotic beats above the mean line may be influenced, but in the majority of experiments they are not. In any case a considerable amount of evidence has been given that the first few dicrotic beats occur at a heightened pressure. In inquiring into the actual cause of the rise, unconscious increase of the rigidity of the limb has to be considered. The regularity of the tracings in the same and different individuals excludes it. Moreover with the weight extension, muscular interference is minimised, and frequently increased rigidity yields a slight fall. An experiment has been devised which places the nature of the rise beyond the shadow of a doubt. The Riva-Rocci armlet is placed on the upper arm and the sphygmograph or finger on the radial pulse. The pressure in the armlet is raised above systolic blood-pressure and a chest compression is performed. The beats immediately pass the block and are recorded or felt at the wrist. The experiment (Figs. 19 A and 21 A, from over 50 similar experiments) is easy to demonstrate and there is no doubt whatever about the result and the conclusion to be drawn from it. Given that the initial rise is due to raised pressure, it still remains to show by a similar method that the dicrotic beats are at a higher pressure than normal. This is more difficult, for the systolic portions of the beats alone force the block and increased dicrotism is not shown.

Exp. 10, Fig. 19 A and B show the records of the right and left pulses taken simultaneously. That from the right arm was taken through a block of 140 mm. Hg the systolic blood-pressure at the time being 135. That the artery was totally occluded at the start was shown from the fact that the lever wrote on a line far below the level of the normal pulse beats. It will be seen that 8 beats have forced the block on the first rise. The tracing then writes a straight line and is again interrupted by beats of the second rise. Beats 6, 7 and 8 show increased dicrotism at a higher pressure than normal. The onset of compression in Fig. 19 B was calculated not only from the time tracing and initial stops on both the papers[1], but by a series of tracings

[1] The stops are not shown in the figure.

similar to that shown in Fig. 20. In tracing A the upper line is a record of the movement of a Hg column into which the subject of experiment blows. In tracing B, the upper line is taken from the movement of the abdominal wall. In all cases the beat taking place at



Figs. 19—21. In Fig. 21 *I*, Inspiration; *C*, Compression; *R*, Release.

the beginning of the chest compression is the first to be affected; and it is affected in whatever phase it happens to be. A number of experiments have been made to estimate the possible rise of blood-pressure

which can take place at the same time as a rise of intrathoracic pressure. Fig. 21 A shows a blood-pressure of 135 mm. Hg raised to over 182[1] mm. Hg by a very forcible expiration with closed glottis, a rise of over 47 mm. Hg. Fig. 21 B is somewhere about the limit. The block is 55 mm. above systolic blood-pressure and the beats are just visible on the tracing, which rises somewhat. It is only the first few beats which pass a block at high pressure, the dicrotic beats are excluded. The line following these beats fails to fall to its original level again. This is due to the nature of the block, which prevents the blood which has entered from again leaving the arm.

Exp. 11. The volume of the limb has been estimated with the plethysmograph in two individuals. Altogether 30 tracings have been taken, at five sittings. Before adjusting the plethysmograph, venous congestion was raised in the limb and maintained throughout each experiment. In the tracing given the plethysmograph style was writing 1 mm. to the left of the style writing from the column of Hg

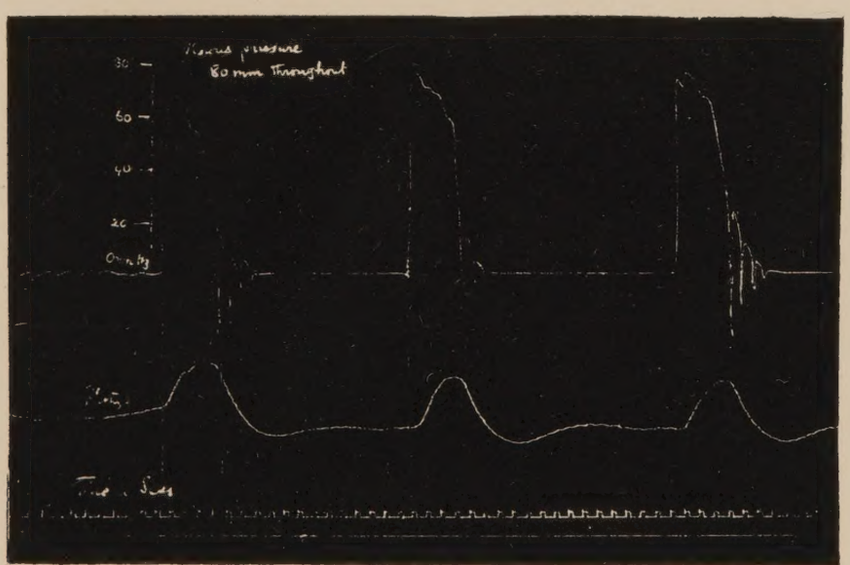


Fig. 22.

and recording the blowing pressure, (the upper line), (Fig. 22). The venous congestion in this instance was 80 mm. throughout in the arm investigated. The blowing pressure is about 76 mm. in each tracing. The record of volume shows a delay of $\frac{1}{2}$—1 second, a period of vaso-dilatation and a period of vaso-constriction following it. The more forcible the compression the smaller is the interval of delay in the rise of the volume curve. Also the more forcible it is, the less is the volume

[1] The armlet was at a pressure of 182 mm. Hg.

plateau prolonged beyond the Hg plateau, though in all cases it extends beyond it. The tracings given are typical of the remainder in every respect but one; the curve indicative of vaso-constriction is frequently more prolonged than is here shown.

That the relations might be demonstrated with greater ease, six Valsalva experiments have been performed on the same individual, the chest compression varying from 58—84 mm. Hg; the Hg curve and volume curve were recorded, and simultaneous tracings were taken from the wrist. The six batches of 3 curves were then compounded to scale, and the result is shown in Fig. 23. The points where the curves cross the base lines are exact, other points are approximate. In the curve

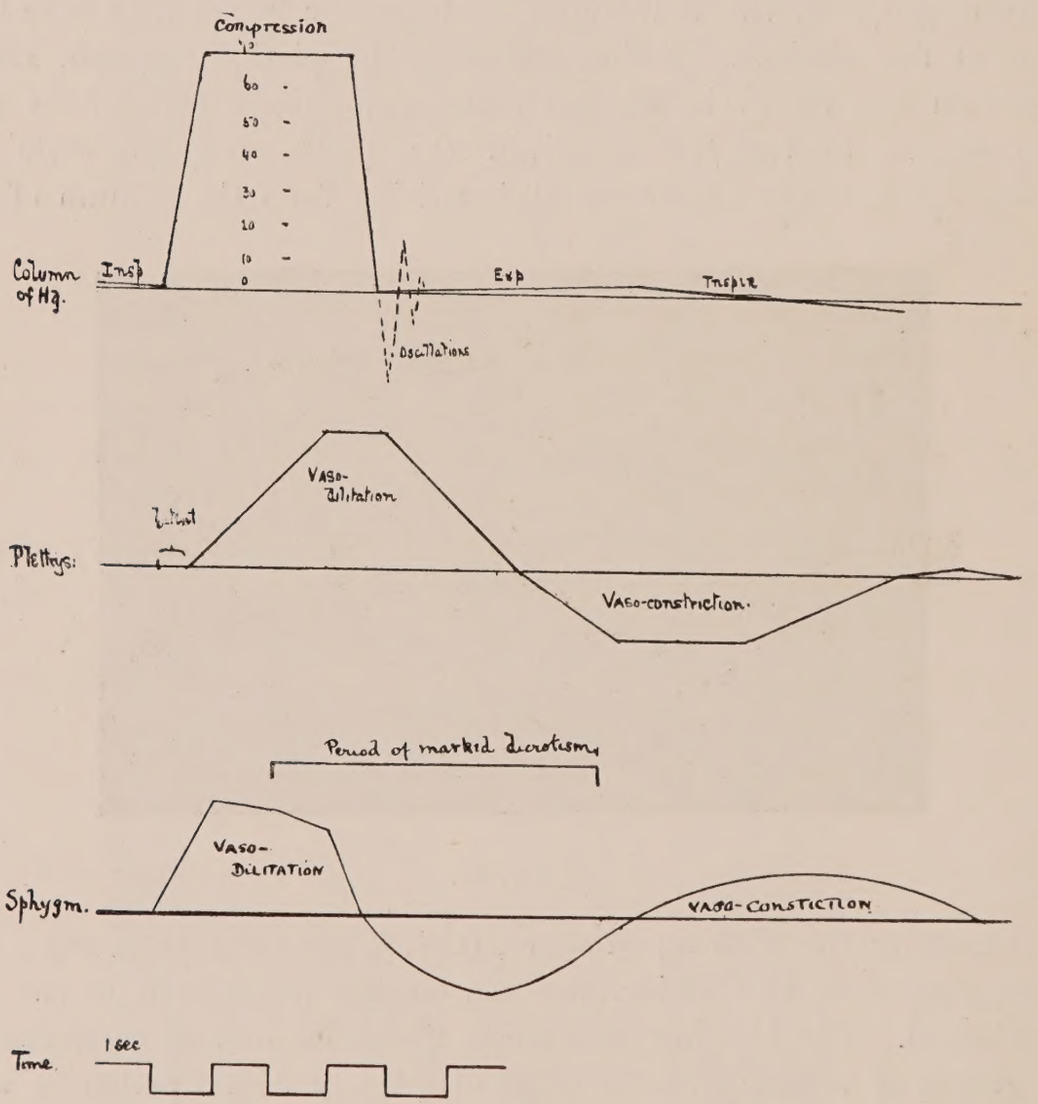


Fig. 23. *Six Valsalva experiments.* Compounded Diagram from careful measurements, giving the relationship of the curves of:

1. Chest compression.
2. Volume of the arm.
3. Pressure in the radial artery as evidenced by the sphygmograph tracing.

taken from the sphygmograph tracings the line joining the bases of the pulse beats was used, as it was found more convenient. The curve taken from the apices is practically identical with it. The diagram shows the relation of dicrotism to volume and pressure. The chief conclusions to be drawn are that dicrotism corresponds neither to increased volume, nor to decreased pressure. It corresponds to those periods when either volume is maximal, or pressure is minimal. The rise in pressure occurs earlier and more rapidly than the increased volume. The second rise of pressure corresponds to the period of vaso-constriction.

The causation of the first rise of pressure. With the first rise of pressure there is an increase in the volume of the limb, though changes in the latter are delayed throughout, and may therefore be secondary to pressure changes. An increase in the volume of the limbs accompanied by a general rise of blood-pressure is well known to accompany contraction of the splanchnic vessels. Reference need only be made to the work of Bayliss[2]. His tracings of volume of the limb in relation to general blood-pressure, as a result of splanchnic stimulation, and the tracings here given are in absolute agreement. They all show the primary delay in the increase of volume, and the more prolonged vaso-constriction following it. The conclusion is that the increased volume of the vessels depends on a greater influx of blood (cp. [21]), that there is a passive distension of the vessels and that the rise of pressure is due to the greater output of blood from the heart consequent upon the rapid emptying of the overfilled lungs into its left side. As the rise in pressure occurs without delay and at any phase of the beat, it is practically certain that some of the blood is derived from the arch of the aorta and great vessels; theoretically there is every reason to believe that this may occur. In this way the immediate rise, and the main rise with the next beat is explainable. It is probable that the outlet for the blood is impeded, during the last stages of the first rise, by venous congestion, but it is not at all certain that this is not counterbalanced by the vaso-dilatation and increased pressure. In animals it has been shown, as mentioned above, that expiratory efforts are accompanied by a fall of pressure after a preliminary rise. This is precisely what occurs in Valsalva's experiment, but the rise is prolonged and the fall is curtailed. The obvious explanation of this is the fact that active compression of the chest is impossible without abdominal rigidity. Such was the explanation of Marey[16], Roy and Adami[21] and others. Experiments on animals differ mainly in this respect, that with the

raised intrathoracic pressure, there is an insufficient rise of abdominal pressure, and the arterial pressure falls. Passive compression of the human chest is said to produce a like result (see Hill, Barnard and Sequeira, *loc. cit.*). The experiments of Zuntz[27] on the breathing of compressed air are in accord with this view. The fall of arterial pressure is generally held to be the result of venous obstruction. I know of no direct evidence to show that a raised intrathoracic pressure interferes with the working capacity of the heart. The nearest approach to such evidence is to be found in the experiments of Cohnheim[4] and the observations of Bolton[3]. The first of these observers impeded the action of the heart by distending the pericardium to a pressure of 230—240 mm. of oil[1], the latter by contracting the pericardium. The fall of arterial pressure in these cases may be due to interference with the veins, and to nervous influences. It is true that a raised intrathoracic pressure will impede the heart in diastole, but it is equally true that it will aid it in systole. In Valsalva's experiment the entry of blood by the inferior vena cava is not interfered with, for it enters under a pressure equal to if not greater than the pressure in the chest. The heart is consequently well fed so long as the splanchnic reservoir lasts. A tracing (Fig. 13) has already been given, which

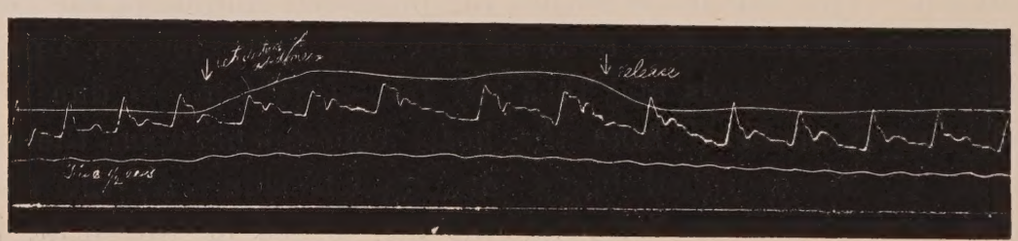


Fig. 24. Retraction of abdomen at 1st, release at 2nd.

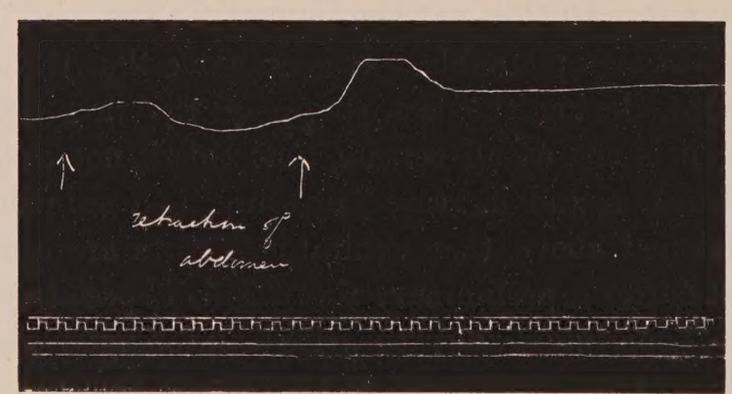


Fig. 25.

[1] Cp. François Franck, *loc. cit.* T. iii. p. 107.

shows the effect of chest compression with the lungs depleted. It is evidence to show that the preliminary part of the raised curve depends mainly on pulmonary blood. Passive or active compression of the abdomen, with quiet breathing gives as is well known a heightened arterial pressure. (For a full account of this subject, see Mummery[17].) An example of the effect of abdominal retraction on the pulse is given in Fig. 24. The effect on the volume of the limb corresponds (Fig. 25).

Exp. 12. Fig. 26 is an example of a pulse record during Valsalva's experiment with a load of 12 stone on the abdomen. The rise in the curve is not so great as might be expected, though the chest compression was to 105 mm. Hg. The fall on release is almost abolished and only two dicrotic beats[1] appear. In a control there was the usual rise and fall and seven dicrotic beats. The inference is that the supply of blood from the abdomen was small, but at high pressure, and that on release of the chest pressure the lungs and not the splanchnic area refilled. The opinion has been stated[2], that increased intrathoracic pressure has little direct effect on the heart muscle. Such increased pressure must be of common occurrence in men engaged in heavy work, in divers, and in crowded streets. Yet syncope under such conditions is a very rare event. The compensating factor, and the protection of the individual, appears to lie in the simultaneous increase of pressure on the splanchnic blood. It is easy to understand that early fatigue of the abdominal

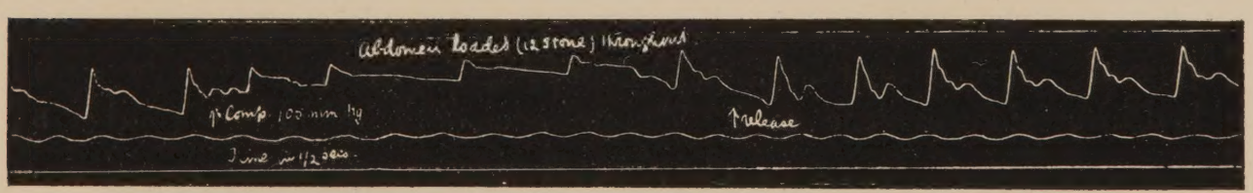


Fig. 26.

muscles, such as might be expected in thoracic breathers, would lead in an individual, compressed in a crowd, to an early development of syncope. The danger of compressing the chest of an anæsthetised patient is well recognised.

The first fall. The curve usually falls a great deal before release. If it falls below the normal line, the beats increase in rapidity. Abolition of the beats has not been met with; the nearest approach to it is shown in Fig. 27; the chest pressure was at 105 mm. Hg. There is a

[1] The beats are not purely dicrotic.

[2] Though it is stated with reserve.

greater tendency to smaller beats when the band fixation is used, also there is the same tendency if from any cause there is venous interference (*i.e.* with light pad pressures, with prolonged compression, and preliminary venous engorgement; cp. Figs. 17 and 18). The tracing is the only one which, during the course of these experiments, showed dicrotism throughout; it is one of the earlier tracings and was probably taken shortly after a trial compression, and before the effects of the first compression were lost. The main fall always occurs on release and is presumably due to refilling of the emptied reservoirs during this period (cp. authors already referred to).

The second rise. The second rise, which is a constant phenomenon, is well seen in Figs. 9 and 19, and is due to vaso-constriction (see Fig. 23) following the passive vaso-dilatation (cp. [23]). The occurrence of this constriction is in accord with the results of Bayliss [2]. The rise of pressure (see Fig. 19) has been calculated (by Exp. 10) at 15—25 mm. Hg. It is not so high as the preliminary rise, though it shows some relation to the force and length of the chest compression. The rise is accompanied by large slow beats, and the subject is aware of a certain amount of palpitation. Bayliss has suggested that the vaso-constriction following passive distension is myogenic in origin. It is possible that these irregular ventricular beats are also myogenic[1].

A fuller discussion of dicrotism is reserved for a future occasion; in the meanwhile stress is laid upon the fact that it may occur at heightened blood-pressures, and that there is no doubt that those observers who attributed the rise of Valsalva's experiment to heightened pressure, were correct in their conclusion, though they seem to have had insufficient evidence for it. The presence of dicrotism in Valsalva's

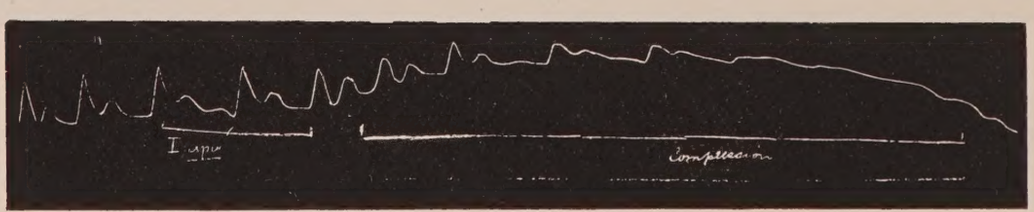


Fig. 27.

experiment, in association with either reduced pressure or increased volume, suggests a definite relationship between volume and pressure as its chief causal factor. This hypothesis would require that any given pressure should have associated with it volumes having a definite

[1] Some recent tracings from the jugular veins by Mackenzie's method showed a regular auricular beat, preceding each ventricular contraction.

minimal limit, for the production of marked dicrotism. There is little evidence in support of the supposition and there is a certain amount of evidence against it. For instance the disappearance of dicrotism at the height of amyl nitrite inhalation effect [12]. There is more reason to suppose that the chief causal factor is that upheld by Galabin [8] (cp. [15] also), Roy and Adami [21], and others, namely, a short, sharp ventricular contraction.

Note on the production of anacrotism.

In some experiments, connected with this research, it has been found that if peripheral resistance be raised by covering the hand with the armlet of the Riva-Rocci, and raising the armlet to a pressure at or about diastolic pressure, the radial pulse of the normal individual becomes markedly anacrotic. The observation is of interest in that it shows that anacrotism may be produced locally and peripherally.

Instances of an anacrotic beat produced distal to an obstruction are given by Sansom [22]. Investigations are now proceeding in this direction[1]. A tracing (Fig. 28) is given illustrating its production in the way

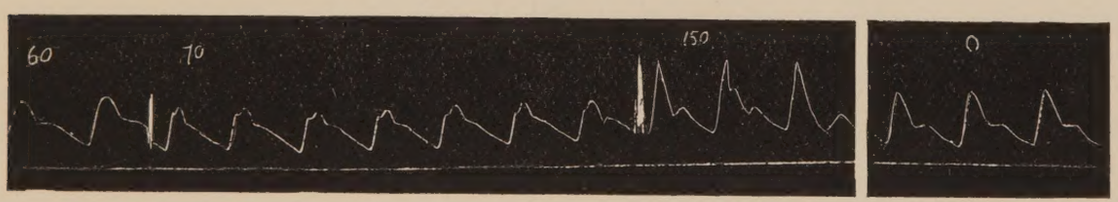


Fig. 28.

described. The pressures in the armlet are marked on the tracing above each section. The diastolic pressure in this individual was 70 mm. Hg.

Chief conclusions.

1. The band used in fixing the Dudgeon's Sphygmograph is a defect in the instrument, when curves indicating pressure are desired. The instrument fitted with weight extension gives a delicate index of pressures.

2. Venous pressure can be approximately measured in the limbs by the combined use of an ordinary Dudgeon's Sphygmograph and a Riva-Rocci apparatus.

[1] Lewis, *Practitioner*, 1906.

3. Venous congestion plays no part in the initial rise of a sphygmograph curve in Valsalva's experiment, if weight extension is used for fixing the instrument. The rise is due to an increased blood-pressure which is capable of easy and certain demonstration. There is simultaneously an increase in the volume of the arterial system of the limbs[1]. The blood is derived mainly from the lungs, partly from the great arteries, and is sustained by increased abdominal pressure. Raised intrathoracic pressure, unless extremely severe, interferes little with the working capacity of the heart.

4. The pressure exerted by the sphygmograph pad, or plethysmograph, can be approximately estimated, by producing venous engorgement with a Riva-Rocci armlet. When the venous pressure reaches this pressure the curve of the tracing is influenced.

5. Dicrotism may be produced in a normal pulse, with a simultaneous rise of blood-pressure. There is no reason to believe that dicrotism is decreased by an increase of blood-pressure *per se*, or that *vice versa* it is increased by a lowering of pressure *per se*[2].

6. The sphygmomanometer armlet serves as a convenient and serviceable plethysmograph when filled with fluid.

7. Anacrotism can be produced with ease and certainty in a normal pulse tracing by increasing peripheral resistance locally to a pressure at or about diastolic pressure.

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[1] The majority of the experiments given can be performed on the leg.

[2] The general teaching of to-day is that dicrotism is increased by a lowered blood-pressure. This view is based on the work of Mahomed[15], Galabin[8], and numerous other observers in this country. The view is supported by the leading text books (Schäfer's *Text Book*[9], Mackenzie[14], Broadbent, *The Pulse*, London, 1890, Sansom[22], and many others). Dicrotism depends not on lowered pressure *per se*, but on those factors which *usually* accompany lowered pressure. In speaking of blood-pressure systolic blood-pressure is referred to.

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